Record: 1

Title: Optimal Vitamin D Status for Colorectal Cancer Prevention: A Quantitative Meta Analysis.

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Holick, Michael F.[6]

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Document Type: Article

Subject Terms: *VITAMIN D
*CALCIUM regulating hormones
*COLON (Anatomy) -- Cancer
*CANCER -- Prevention

Abstract: Background: Previous studies, such as the Women's Health Initiative, have shown that a low dose of vitamin D did not protect against colorectal cancer, yet a meta-analysis indicates that a higher dose may reduce its incidence. Methods: Five studies of serum 25(OH)D in association with colorectal cancer risk were identified using PubMed. The results of all five serum studies were combined using standard methods for pooled analysis. The pooled results were divided into quintiles with median 25(OH)D values of 6, 16, 22, 27, and 37 ng/mL. Odds ratios were calculated by quintile of the pooled data using Peto's Assumption-Free Method, with the lowest quintile of 25(OH)D as the reference group. A dose–response curve was plotted based on the odds for each quintile of the pooled data. Data were abstracted and analyzed in 2006. Results: Odds ratios for the combined serum 25(OH)D studies, from lowest to highest quintile, were 1.00, 0.82, 0.66, 0.59, and 0.46 (p _{trend}<0.0001) for colorectal cancer. According to the DerSimonian-Laird test for homogeneity of pooled data, the studies were homogeneous (chi²=1.09, df=4, p=0.90. The pooled odds ratio for the highest quintile versus the lowest was 0.49 (p<0.0001, 95% confidence interval, 0.35–0.68). A 50% lower risk of colorectal cancer was associated with a serum 25(OH)D level ≥33 ng/mL,

compared to ≤12 ng/mL. Conclusions: The evidence to date suggests that daily intake of 1000–2000 IU/day of vitamin D₃ could reduce the incidence of colorectal with minimal risk. [Copyright &y& Elsevier]

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Accession Number: 23972571

Database: Academic Search Premier

Record: 1

Title: Ultraviolet B Irradiance and Incidence Rates of Bladder Cancer in 174 Countries.

Authors: Mohr, Sharif B.[1,2]
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Grant, William B.[3]
Garland, Frank C.[1,2]

Source: American Journal of Preventive Medicine; Mar2010, Vol. 38 Issue 3, p296-302, 7p

Document Type: Article

Subject Terms: *ULTRAVIOLET radiation -- Physiological effect
*MULTIPLE regression analysis
*SMOKING
*MEDICAL care
*PUBLIC health
*BLADDER
*CANCER
DISEASE incidence

NAICS/Industry Codes525120 Health and Welfare Funds

Abstract: Background: Although nearly half of bladder cancer cases are due to smoking, the cause of nearly half is unexplained. Purpose: This study aims to determine whether an inverse association exists between ultraviolet B (UVB) irradiance and incidence rates of bladder cancer worldwide. Methods: This study used an ecologic approach. Age-adjusted incidence rates of bladder cancer from 2002 were obtained for all 174 countries in GLOBOCAN, a database of the International Agency for Research on Cancer. The relationship of latitude and estimated serum 25-hydroxyvitamin D [25(OH)D] with incidence rates was determined. The independent contributions to incidence rates of bladder cancer of UVB, per capita cigarette consumption in 1980, and per capita health expenditure for 2001 were assessed using multiple regression. The analyses were performed in July 2008. Results: Bladder cancer incidence rates were higher in countries at higher latitudes than those nearer to the equator ($r=-0.66$, 95% CI=−0.74, −0.57, $p<0.01$). Ultraviolet B irradiance was independently inversely associated with incidence rates of bladder cancer after controlling for per capita cigarette consumption ($\beta=-0.28$, 95% CI=−0.51, −0.05; R ² for model=0.38, $p<0.0001$). Further, UVB irradiance was also inversely associated with incidence rates after

controlling for per capita health expenditure (β=−0.23, 95% CI=−0.36, −0.01; R ² for model=0.49, p<0.0001) in a separate regression model. Conclusions: Further investigation is needed to confirm the associations identified in this study using observational studies of individuals. The focus of this research should include the association of serum 25(OH)D levels with risk of bladder cancer. [Copyright &y& Elsevier]

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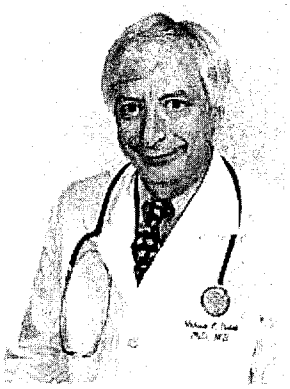
Life Extension Magazine September 2010

BOOK
EXCERPT

The Vitamin D Solution

By Michael F. Holick, PhD, MD

If I had to give you a single secret ingredient that could apply to the prevention—and treatment, in many cases—of heart disease, common cancers, stroke, infectious diseases from influenza to tuberculosis, type 1 and 2 diabetes, dementia, depression, insomnia, muscle weakness, joint pain, fibromyalgia, osteoarthritis, rheumatoid arthritis, osteoporosis, psoriasis, multiple sclerosis, and hypertension, it would be this: vitamin D.



I have been traveling around the world not only lecturing about vitamin D but also hearing from physicians how common vitamin D deficiency is. It's not only the most common nutritional deficiency in the world, but it's also the most common medical condition, affecting at least one billion people. Three out of every four Americans are deficient in vitamin D, up from one out of two twenty years ago.

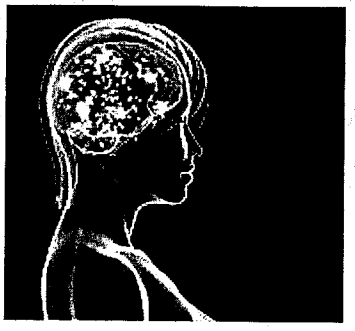
A HORMONE, NOT A VITAMIN

Naturally, we're disposed to think about vitamin D as a vitamin—a substance that we get from our diets, like vitamin C or niacin, and that participates in biological reactions to help the body operate optimally. But despite its name, vitamin D isn't really a vitamin. Vitamin D is in a class by itself; its far-reaching effects on the body are aligned with how hormones act to influence metabolic pathways, cellular functions, and the expression of myriad genes. Vitamin D's active metabolic product in the body, in fact, is a molecule called 1,25-dihydroxyvitamin D (let's call it 1,25-vitamin D for simplicity), which is a secosteroid hormone that directly or indirectly targets more than two thousand genes, or about 6 percent of the human genome.

FROM BONE HEALTH TO BRAIN HEALTH

Contrary to what was previously believed—that vitamin D receptors were only in bones, intestines, and kidneys—we now know that vitamin D receptors are *everywhere* in the body. There is even proof that vitamin D receptors exist in the brain and that the active form of vitamin D stimulates the production of mood-elevating serotonin. This explains how it may help reduce depression (or just a chronically foul mood). Fat cells, too, have vitamin D receptors, and fat cells can be more metabolically active (burn more calories) if they have more vitamin D. People tend to think that fat cells are like inanimate blobs of lard when in fact they are active participants in the process by which your brain learns that you're full and don't need to take another bite of food. When you've had enough, fat cells secrete a hormone called leptin that allows you to push away from the table. A lack of vitamin D will interfere with this appetite-suppressing hormone whose job it is to regulate your body weight. And we all know what an unchecked appetite can lead to: weight gain and a higher risk of developing type 2 diabetes. Speaking of which, vitamin D deficiency has also been shown to exacerbate type 2 diabetes, impair insulin production in the pancreas, and increase insulin resistance.

BENEFITS OF VITAMIN D IN BRIEF



Bone health: prevents osteopenia, osteoporosis, osteomalacia, rickets, and fractures.

Cellular health: prevents certain cancers, such as prostate, pancreatic, breast, ovarian, and colon; prevents infectious diseases and upper-respiratory-tract infections, asthma, and wheezing disorders.

Organ health: prevents heart disease and stroke; prevents type 2 diabetes, periodontitis and tooth loss, and other inflammatory diseases.

Muscular health: supports muscle strength.

Autoimmune health: prevents multiple sclerosis, type 1 diabetes mellitus, Crohn's disease, and rheumatoid arthritis.

Brain health: prevents depression, schizophrenia, Alzheimer's disease, and dementia.

Mood-related health: prevents seasonal affective disorder, premenstrual syndrome (PMS, also known as premenstrual tension), and sleeping disorders.

VITAMIN D AND OBESITY

Because vitamin D is stored in fat cells, you'd think that people with excess fat would have plenty of extra vitamin D on hand to make up any shortage. As it turns out, that thinking is wrong, and a parallel relationship exists between vitamin D deficiency and obesity. The fatter you are, the higher your risk for a deficiency. Why? The vitamin D essentially gets locked inside the fat cells, unavailable for use.

In one of my studies, we exposed obese and nonobese individuals to the same amount of UVB radiation and showed that obese people can only raise their blood levels of vitamin D by about 45 percent compared to a normal-weight person. Obese people (defined as those with a body mass index, or BMI, above 30) often need at least twice as much vitamin D to satisfy their body's needs. With the majority of Americans overweight or obese these days, it's not a stretch to understand why a similar number of people are vitamin D deficient. The two epidemics have worsened in unison.

WHAT CONSTITUTES A DEFICIENCY?

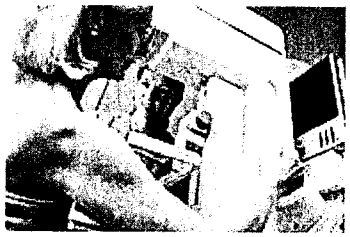
My extensive studies have helped redefine what it means to be vitamin D deficient. Before one of my publications in the *Lancet* in 1998, vitamin D deficiency was defined as a having 25-vitamin D levels below **10** nanograms per milliliter. I demonstrated, however, that a blood level of twice that—20 nanograms per milliliter—is needed to prevent an unhealthy elevation in parathyroid hormone level, a sign of vitamin D deficiency.

Obesity-related conditions now account for nearly 10 percent of all medical spending, having doubled in the last decade. It's hard to believe that the obesity rate could rise 37 percent in just eight years alone, but that's exactly what happened between 1998 and 2006—bringing a breathtaking one third of the adult American population into the obese camp. Why the spike in such a short time frame?

We've evolved to store vitamin D in fat. This allows us to have ample supplies on hand during the long winter months, when it's virtually impossible to make vitamin D—when the active synthesis of vitamin D from the sun is in hibernation until the spring. But humans did not evolve to carry such copious amounts of excessive fat. The result is that this fat begins to have negative effects on the body's metabolism and hormonal balance.

Contrary to what you might think, overweight people don't have higher levels of 25-vitamin D due to their higher fat content. They have lower levels, because the excess fat absorbs and holds onto the vitamin D so that it cannot be used for bone building and cellular health. Unlike a normal-weight person, whose fat is continually being recycled so the vitamin D can be released, those with relatively immobile fat stores cannot access their vitamin D, which is literally locked up in their adipose tissue. Making matters worse, obese people are frequently vitamin D deficient to start with because they go outside much less. Obese people need two to three times more vitamin D a day than those of normal weight, so I advise such patients to take between 3,000 and 6,000 IU of vitamin D a day.

CRACKING ONE OF CANCER'S CODES BREAST CANCER



Here's a staggering statistic: Women who are deficient in vitamin D at the time they are diagnosed with breast cancer are nearly 75 percent more likely to die from the disease than women with sufficient vitamin D levels. What's more, their cancer is twice as likely to metastasize to other parts of the body.

In the United States, more than forty thousand women die from breast cancer every year—making it the deadliest killer of women after heart disease. One woman in eight either has or will develop breast cancer in her lifetime. There are 214,000 new cases and 41,000 deaths from breast cancer each year in the United States. A 2008 study found that women who had a vitamin D deficiency at the time they were diagnosed with breast cancer were 94 percent more likely to have their cancer spread than women with adequate 25-vitamin D levels in their bodies.

In May 1999, a landmark study by Dr. Esther John, based on the meticulous analysis of breast cancer statistics from the

National Health and Nutrition Examination Survey, was published. The authors conclude definitively that sun exposure and a vitamin D–rich diet significantly lower the risk of breast cancer.

PROSTATE CANCER

Only heart attacks and lung cancer kill more men than cancer of the prostate. Cancer of the prostate is especially feared by men because surgical treatment for this form of cancer frequently results in impotence. A study in the August 2001 issue of the *Lancet* proves that the risk of developing prostate cancer is directly related to sunlight exposure. The study divided people into four groups according to how much sunlight they had been exposed to. The lowest quarter, or quartile, of the study participants were three times more likely to develop prostate cancer than those in the highest quartile of sun exposure. The results show that those in the highest quartile reduced their risk of developing prostate cancer by 66 percent. Those in the second and third quartiles also had a significantly lower chance of getting prostate cancer compared with those in the lowest quartile, who received the least sun exposure. Another study took a long look, over almost two years, at men with prostate cancer who received 2,000 IU of vitamin D a day and found that overall the men had a 50 percent reduction in the rise of their levels of prostate specific antigen (PSA), which is an indicator of prostate cancer activity.

COLON CANCER

Cancer of the colon and its neighboring area, known sometimes as colorectal cancer, affects both men and women. Like breast cancer and prostate cancer, colorectal cancer is seen much more frequently than skin cancers and is much more deadly. About 150,000 Americans are told each year that they have colon cancer, and about 35 percent of these will die of it.

A study published in the *Journal of Clinical Oncology* in 2008, conducted by lead researcher Dr. Kimmie Ng of the Dana-Farber Cancer Institute in Boston found that high blood levels of 25-vitamin D increased colon cancer patients' survival rate by 48 percent. In this study, Dr. Ng and her team collected data on 304 patients who had been diagnosed with colon cancer between 1991 and 2002. Everyone in the study had had their 25-vitamin D blood levels measured a minimum of two years before being diagnosed with the disease. The patients were tracked until they died or until the study ended in 2005; 123 patients died, 96 of them from colon or rectal cancer during the follow-up period. Dr. Ng and her team found that the patients with the highest 25-vitamin D levels were 39 percent less likely to die from colorectal cancer than the patients who had the lowest levels.

These findings are consistent with dozens and dozens of other observations that have been made in the past decade, including those by Dr. Cedric Garland. His lab reports that you are three times less likely to die from colon cancer if you have healthy levels of 25-vitamin D in your bloodstream.

A NEW MODEL FOR CANCER?

Though it's a stretch to say vitamin D can totally prevent and cure cancer, some scientists have been bold enough to suggest a whole new theory about cancer. Just last year, the Garland brothers raised the possibility that there's another story behind cancer's genesis in the body. The current scientific model assumes that a genetic mutation is cancer's point of origin. But what if that assumption is wrong? What if there is another way to explain how cancer develops? Those are the questions the Garlands put forth, which were published in the *Annals of Epidemiology* and immediately picked up by the media.

First, Dr. Cedric Garland and his team pointed to a host of research that suggests cancer develops when cells lose the ability to stick together in a healthy, normal way. He went on to argue that the key factor in this initial triggering of a malignancy could well be a lack of vitamin D. According to Dr. Garland, researchers have documented that with enough activated vitamin D present, cells adhere to one another in tissue and act as normal, mature cells. But if there is a deficiency of activated vitamin D, cells can lose this stick-to-each-other quality, as well as their identity as differentiated cells. The result? They may revert to a dangerous, immature state and become cancerous.

What can stop this process from occurring, says Dr. Garland, is ample supplies of vitamin D in the body. Whether or not this theory can be proven true will be told by future studies and research.

TESTING, TESTING, 1-2-3

The only surefire way to know for certain the extent of your vitamin D deficiency is to ask for a *25-hydroxyvitamin D* test, also called a 25(OH) D test. This is the circulating form of vitamin D that the liver generates and that then becomes activated by the kidneys. While it's intuitive to think you'd want to test for the body's "active form" rather than just a precursor, testing for the activated vitamin D (1,25-vitamin D) does not give an accurate portrayal of your vitamin D status.

And here's the rub: many doctors order the wrong test, and when the results come back showing a normal level of activated vitamin D, they think everything is D-okay. You could, however, be suffering from a serious deficiency even though your activated levels appear normal—or even elevated.

FOLLOWING DR. HOLICK'S RECOMMENDATIONS

Dr. Holick uses vitamin D supplements, milk, and sensible sun exposure to keep his ***25-hydroxyvitamin D*** blood levels at **50** ng/mL. Dr. Holick believes that this and higher levels of 25-hydroxyvitamin are optimal.

Life Extension® has always used the more accurate ***25-hydroxyvitamin D*** blood test recommended by Dr. Holick. Foundation members can order this ***25-hydroxyvitamin* D blood test** for **$47** by calling **1-800-208-3444.**

To order a copy of *The Vitamin D Solution* by Dr. Michael F. Holick, call 1-800-544-4440 or visit www.LifeExtension.com Item#33824 • Retail price $25.95 Member price $18.17

Record: 1

Title: Intake of vitamin D and risk of type 1 diabetes: a birth-cohort study.

Authors: Hypponen, Elina
Laara, Esa
Reunanen, Antti
Jarvelin, Marjo-Riitta
Virtanen, Suvi M

Source: Lancet; 11/3/2001, Vol. 358 Issue 9292, p1500, 4p, 2 Charts

Document Type: Article

Subject Terms: *VITAMIN D
*DIABETES
*DIETARY supplements
*MEDICINE, Preventive

Abstract: Summary: Background: Dietary vitamin D supplementation is associated with reduced risk of type 1 diabetes in animals. Our aim was to ascertain whether or not vitamin D supplementation or deficiency in infancy could affect development of type 1 diabetes. Methods: A birth-cohort study was done, in which all pregnant women (n=12 055) in Oulu and Lapland, northern Finland, who were due to give birth in 1966 were enrolled. Data was collected in the first year of life about frequency and dose of vitamin D supplementation and presence of suspected rickets. Our primary outcome measure was diagnosis of type 1 diabetes by end of December, 1997. Findings: 12 058 of 12 231 represented live births, and 10 821 (91% of those alive) children were followed-up at age 1 year. Of the 10 366 children included in analyses, 81 were diagnosed with diabetes during the study. Vitamin D supplementation was associated with a decreased frequency of type 1 diabetes when adjusted for neonatal, anthropometric, and social characteristics (rate ratio [RR] for regular vs no supplementation 0.12, 95% CI 0.03-0.51, and irregular vs no supplementation 0.16, 0.04-0.74. Children who regularly took the recommended dose of vitamin D (2000 IU daily) had a RR of 0.22 (0.05-0.89) compared with those who regularly received less than the recommended amount. Children suspected of having rickets during the first year of life had a RR of 3.0 (1.0-9.0) compared with those without such a suspicion. Interpretation: Dietary vitamin D supplementation is associated with reduced risk of type 1 diabetes. Ensuring adequate vitamin D supplementation for infants could help to reverse the increasing trend in the incidence of type 1 diabetes. [ABSTRACT FROM AUTHOR]

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Record: 1

Title: Vitamin D and Calcium Intake in Relation to Type 2 Diabetes in Women.

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Willett, Walter C.[3]
Manson, Joann E.[3,4]
Hu, Frank B.[3]

Source: Diabetes Care; Mar2006, Vol. 29 Issue 3, p650-656, 7p, 4 Charts

Document Type: Article

Subject Terms: *VITAMIN D
*CALCIUM
*NON-insulin-dependent diabetes
*DIABETES
*ENDOCRINE glands -- Diseases

Abstract: OBJECTIVE -- The purpose of this study was to prospectively examine the association between vitamin D and calcium intake and risk of type 2 diabetes. RESEARCH DESIGN AND METHODS -- In the Nurses' Health Study, we followed 85,779 women who had no history of diabetes, cardiovascular disease, or cancer at baseline for the development of type 2 diabetes. Vitamin D and calcium intake from diet and supplements was assessed every 2-4 years. During 20 years of follow-up, we documented 4,843 incident cases of type 2 diabetes. RESULTS -- After adjusting for multiple potential confounders, there was no association between total vitamin D intake and type 2 diabetes. However, the relative risk (RR) of type 2 diabetes was 0.87 (95% CI 0.75-1.00; P for trend = 0.04) comparing the highest with the lowest category of vitamin D intake from supplements. The multivariate RRs of type 2 diabetes were 0.79 (0.70-0.90; P for trend <0.001) comparing the highest with the lowest category of calcium intake from all sources and 0.82 (0.72-0.92; P for trend <0.001) comparing the highest with the lowest category of calcium intake from supplements. A combined daily intake of >1,200 mg calcium and >800 IU vitamin D was associated with a 33% lower risk of type 2 diabetes with RR of 0.67 (0.49-0.90) compared with an intake of <600 mg and 400 IU calcium and vitamin D, respectively. CONCLUSIONS -- The results of this large prospective study suggest a potential beneficial role for both vitamin D and calcium intake in reducing the risk of type 2 diabetes. [ABSTRACT FROM AUTHOR]

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Full Text Word Count: 5731

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Record: 1

Title: Seasonal distribution of acute myocardial infarction in the second National Registry of Myocardial Infarction.

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Source: Journal Of The American College Of Cardiology [J Am Coll Cardiol] 1998 May; Vol. 31 (6), pp. 1226-33.

Publication Type: Journal Article; Research Support, Non-U.S. Gov't

Language: English

Journal Information: *Country of Publication:* UNITED STATES *NLM ID:* 8301365 *Publication Model:* Print *Cited Medium:* Print *ISSN:* 0735-1097 (Print) *Linking ISSN:* 07351097 *NLM ISO Abbreviation:* J. Am. Coll. Cardiol. *Subsets:* Core Clinical (AIM); MEDLINE

MeSH Terms: Registries*
Seasons*
Myocardial Infarction/*epidemiology
Aged; Female; Hospitalization; Humans; Male; Middle Aged; Myocardial Infarction/etiology; United States/epidemiology

Abstract: **Objectives:** This observational study sought to determine whether cases of acute myocardial infarction (AMI) reported to the second National Registry of Myocardial Infarction (NRMI-2) varied by season.

Background: The existence of circadian variation in the onset of AMI is well established. Examination of this periodicity has led to new insights into pathophysiologic triggers of atherosclerotic plaque rupture. Although a seasonal pattern for mortality from AMI has been previously noted, it remains unclear whether the occurrence of AMI also displays a seasonal rhythmicity. Documentation of such a pattern may foster investigation of new pathophysiologic determinants of plaque rupture and intracoronary thrombosis.

Methods: We analyzed the number of cases of AMI reported to NRMI-2 by season during the period July 1, 1994 to July 31, 1996. Data were normalized so that seasonal occurrence of AMI was reported according to a standard 90-day length.

Results: A total of 259,891 cases of AMI were analyzed during the study period. Approximately 53% more cases were reported in winter than during the summer. The same seasonal pattern (decreasing occurrence of

reported cases from winter to fall to spring to summer) was seen in men and women, in different age groups and in 9 of 10 geographic areas. In-hospital case fatality rates for AMI also followed a seasonal pattern, with a peak of 9% in winter.

Conclusion: The present results suggest that there is a seasonal pattern in the occurrence of AMIs reported to NRMI-2 that is characterized by a marked peak of cases in the winter months and a nadir in the summer months. This pattern was seen in all subgroups analyzed as well as in different geographic areas. These findings suggest that the chronobiology of seasonal variation in AMI may be affected by variables independent of climate.

Comments: Comment in: J Am Coll Cardiol. 1999 Jun;33(7):2088-9. (PMID: 10362222)
Comment in: J Am Coll Cardiol. 1998 Dec;32(7):2103-4. (PMID: 9857903)

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